# Research

# Human Health Benefits from Fish Consumption vs. Risks from Inhalation Exposures Associated with Contaminated Sediment Remediation: Dredging of the Hudson River

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**BACKGROUND:** Billions of dollars are spent on environmental dredging (ED) to remediate contaminated sediments, with one goal being reduced human health risks. However, ED may increase health risks in unanticipated ways, thus potentially reducing net benefits.

**OBJECTIVES:** To assess the ways that ED may increase health risks in unanticipated ways, thus potentially reducing net benefits, we quantitatively assessed a subset of population health benefits and risks of ED, using the 2009–2015 remediation of the Hudson River Polychlorinated Biphenyls (PCBs) Superfund Site as a case study. Three remediation scenarios were evaluated: No Action (NA), Source Control (SC), and ED.

**METHODS:** We quantified health benefits for each scenario from reduced PCB levels in Hudson River fish, and health risks from ED operations due to increased inhalation exposures to PCBs and fine particulate matter ( $PM_{2.5}$ ), using disability-adjusted life years (DALYs) as a common metric. Occupational health risks were also considered in a separate sensitivity analysis. Estimates of population-level benefits and risks included Monte Carlo simulation-based uncertainty analysis.

**RESULTS:** Under NA, fish consumption would result in an estimated health burden of 112 DALYs, and ED would lead to a reduction of 15 DALYs in excess of SC. ED operations were estimated to induce a total burden of 33 DALYs, dominated by  $PM_{2.5}$  impacts from rail transport emissions (32 DALYs). Including uncertainty, the net health benefit of ED ranged from -138 to +1,326 avoided DALYs (90% confidence), with a median of -11 avoided DALYs.

**CONCLUSIONS:** For the considered impacts, ED in the Hudson River might not have led to an overall net positive human health impact. The benefits and risks of ED, however, have different degrees of uncertainty and involve different populations. Reducing long-distance transport of dredged sediment is a priority. This comparative approach could be used prospectively to better determine trade-offs involved in different remediation scenarios and to improve remediation design to maximize benefits while minimizing risks. https://doi.org/10.1289/EHP5034

# Introduction

Many surface waters throughout the United States have sediments contaminated with legacy pollutants from former industrial, mining, and/or agricultural activities (U.S. EPA 2004a). Some of these chemicals can bioaccumulate in aquatic organisms, including fish consumed by local anglers and their families. Depending on the chemical, its concentration and bioaccumulation potential, and the frequency and duration of exposure, these individuals can be at risk of developing adverse health outcomes. Thus, the potential negative effects of contaminated sediments on human health are compelling reasons to consider remediating them (NRC 2007).

Removal of sediments by dredging is one commonly used, yet costly, method of remediation. It is unclear, however, to what extent this remedy is directly responsible for reduced human health risk, with a primary reason being shortcomings in available monitoring data (NRC 2007). There is also a need to comprehensively investigate the trade-offs of dredging in terms of health impacts beyond those associated with the reduced sediment concentrations that typically drive these assessments. For instance, health benefits from reduced sediment concentrations may be partly offset by resuspension of sediment and release of contaminants into the water column during dredging (Bridges et al. 2010). Additionally, environmental dredging (ED) is a highly complex industrial operation that itself involves potential health risks. For example, dredging and transportation equipment can lead to emission and/or formation of considerable amounts of primary and secondary fine particulate matter (PM<sub>2.5</sub>) as well as nitrogen oxides (NO<sub>x</sub>) (Anderson 2008; U.S. EPA 2004b). Ambient PM2.5 is an important contributor to cardiovascular and respiratory diseases, including lung cancer (Brook et al. 2010; IARC 2013; Lepeule et al. 2012; Pope and Dockery 2006). Dredging projects may also expose workers to chemical and physical hazards. In sum, appropriate risk assessments of any remediation strategy, especially dredging, should consider and compare respective risks and benefits.

The Hudson River Polychlorinated Biphenyls (PCBs) Superfund Site (the Site) provides a promising case study for evaluating trade-offs between health risks and benefits of ED. Approximately 200 river-miles long, the Site consists of the Upper Hudson (approximately 40 river-miles between Hudson Falls and the Federal Dam at Troy, New York) and the Lower Hudson (approximately 160 river-miles between the Federal Dam at Troy and Battery Park). From 1947 to 1977, two General Electric capacitor-manufacturing facilities discharged an estimated 1.3 million pounds of PCBs into the Upper Hudson (U.S. EPA 2017). PCBs are carcinogenic to humans (IARC 2016), and

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consistent positive associations have been observed in epidemiological studies for a variety of noncancer adverse health outcomes (Faroon and Olson 2000; Faroon and Ruiz 2016). Between 2009 and 2015, with an off-season in 2010, a dredging remedy costing \$1.7 billion USD (Haggard 2017) removed approximately 2.65 million cubic yards of PCB-contaminated sediment from the Upper Hudson (U.S. EPA 2017). Extensive data sets of PCB measurements in fish, sediment, water, and air are available (U.S. EPA 2017), complemented by mechanistic modeling of PCB concentrations in various media (TAMS Consultants et al. 2000a, 2000b). Furthermore, rigorous documentation and monitoring has generated a wealth of relevant information.

Focusing on the Site, this study sought to extensively evaluate and compare the potentially avoided and induced population health burden of this major ED project. We first evaluated overall human health impacts associated with fish consumption under three scenarios: No Action (NA), Source Control (SC), and the selected remedy combining SC with ED (SC&ED). The details of these scenarios are summarized in Table 1. In each case, we estimated the potential cancer and noncancer health burden on recreational anglers and their families due to consumption of PCB-contaminated fish. These risks constituted the basis for response action for the Site (U.S. EPA 2002a). For ED, we also estimated the health burden induced by this operation from increased air emissions of PCBs and from primary and secondary PM<sub>2.5</sub>. To enable comparison of benefits and risks involving different health effects, we expressed health impacts in terms of disability-adjusted life years (DALYs), representing the number of healthy years of life lost in a population accounting for premature death and disability (Forouzanfar et al. 2016). Finally, we conducted a separate sensitivity analysis estimating the potential health burden of ED on project workers due to inhalation exposures and fatal incidents (physical hazards).

#### Methods

#### **General Framework**

The general framework of our analysis assesses the average damage and associated long-term net health benefit under each scenario as the sum of several individual components. First, the induced health burden from consumption of Hudson River fish ( $\rm IB_{fish,PCB}$ ) was calculated under each scenario. In this paper, "fish" represents both fish and shellfish/crabs. Then, the "avoided burden" of SC ( $\rm AB_{SC}$ ) was represented by the difference between the induced burdens under SC and NA. Similarly, the avoided burden of ED was represented by the difference between the induced burdens under SC&ED and SC:

$$AB_{SC} = IB_{fish,PCB,NA} - IB_{fish,PCB,SC}$$
(1)

$$AB_{ED} = IB_{fish,PCB,SC} - IB_{fish,PCB,SC\&ED}$$
(2)

The benefits of ED from reduced PCB levels in fish were then offset by the burden induced by the ED operation ( $IB_{ED}$ ). For this, we focused on risks to surrounding communities due to increased air emissions of PCBs ( $IB_{air,PCB,communities}$ ) and on risks to the regional and U.S. populations (including surrounding communities) due to air emissions of primary and secondary PM<sub>2.5</sub> from diesel-powered equipment ( $IB_{PM_{2.5},general population}$ ). The total induced health burden was then calculated as follows:

$$IB_{ED} = IB_{air,PCB,communities} + IB_{PM_{2.5},general population}$$
 (3)

The net health benefit of ED was represented by the difference between avoided and induced burdens:

Net health benefit<sub>ED</sub> = 
$$AB_{ED} - IB_{ED}$$
 (4)

Equations 3 and 4 assume that the induced burden of ED on project workers is negligible; i.e., their counterfactual scenario in the absence of dredging would be to work in jobs with similar risk. As a separate sensitivity analysis, we conducted a fully attributional assessment that quantified potential health risks of ED to project workers due to increased inhalation of PCBs and PM<sub>2.5</sub> (IB<sub>air,PCB,workers</sub>), and IB<sub>PM2.5</sub>,workers), and fatal occupational incidents (IB<sub>fatal incidents,workers</sub>), yielding an upper-bound induced health burden:

 $IB_{ED,sensitivity} = IB_{air,PCB,communities} + IB_{PM_{2.5},general \, population}$ 

+ IB<sub>air,PCB,workers</sub> + IB<sub>PM2.5</sub>,workers + IB<sub>fatal incidents</sub>,workers

The effective marginal induced health burden of ED would likely fall within these two bounds (i.e., between  $IB_{ED}$  and  $IB_{ED,sensitivity}$ ).

The methods for calculating each of these components are further described below. Table 2 and Table S1 summarize the primary model input parameters for all considered health impact pathways. Assuming mutual independence among these parameters, we accounted for both variability and uncertainty using Monte Carlo simulation implemented in R (version 3.4; R Development Core Team 2017). For some parameters, we adapted an approach from MacLeod et al. (2002), assuming log-normal probability distributions. Table 3 summarizes these distributions, and the Supplemental Material (Tables S2–S4) summarizes the remaining input data used in the uncertainty analyses.

 Table 1. Remediation scenarios for the Hudson River PCBs Superfund Site (U.S. EPA 2002a).

Scenario (Abbreviation)	Notes		
No Action (NA)	• No active remediation or source control.		
	• Incorporates existing institutional controls, notably fish consumption advisories for the Lower Hudson administered by the		
	New York State Department of Health, and a fish consumption ban for the Upper Hudson administered by the New York		
	State Department of Environmental Conservation.		
	• The U.S. EPA's baseline exposure assumptions did not include institutional controls for this scenario. (TAMS Consultants		
	and Gradient Corporation 2000.)		
Source Control (SC)	• Incorporates existing institutional controls as under NA.		
	• Assumes a separate source control action near the General Electric Hudson Falls plant, reducing the upstream contribution		
	from an average of $0.16 \text{ kg}_{\text{PCB}}/\text{d}$ to an average of $0.0256 \text{ kg}_{\text{PCB}}/\text{d}$ on 1 January 2005.		
	• Relies on naturally occurring attenuation processes (e.g., biodegradation, biotransformation, bioturbation, diffusion,		
	dilution, adsorption, volatilization, chemical reaction or destruction, resuspension, downstream transport, and burial by		
	cleaner materials) to reduce concentrations of PCBs in Hudson River sediments.		
Selected remedy of Source	• Incorporates existing institutional controls as under NA.		
Control with	• Assumes the same upstream source control action as SC.		
Environmental Dredging	• Includes targeted environmental dredging in the Upper Hudson under a 6-y implementation timeframe.		
(SC&ED)	• Assumes a 0.13% release rate (resuspension) of Tri + PCBs at the dredge head.		

Table 2. Summary of primary model parameters for all considered health impact pathways.

Parameter	Unit	Value
Fish tissue PCB concentration $[\overline{C(t)}_{fish, PCB}]$ : Upper; Lower Hudson		
NA scenario	$mg_{PCB}/kg_{fish}$	0.825; 0.355
SC scenario	$mg_{PCB}/kg_{fish}$	0.424; 0.218
SC&ED scenario	$mg_{PCB}/kg_{fish}$	0.273; 0.184
Individual fish ingestion rate $(\overline{IR}_p)$		
Twice per year	kg <sub>fish</sub> /person-y	0.5
Twice per month	kg <sub>fish</sub> /person-y	4
Twice per week	kg <sub>fish</sub> /person-y	17
Fish PCB cancer dose–response $(DR_{ADD_n \text{ fish PCB},e})$	cases/kg <sub>PCB intake</sub>	0.6
Fish PCB noncancer dose–response $(DR_{ADD_n \text{ fish}, PCB, e})$	cases/kg <sub>PCB intake</sub>	See Figure S2
PCB cancer severity factor $(SF_{PCB,e})$	DALY/case	4.3
PCB noncancer severity factor (SF <sub>PCB,e</sub> )	DALY/case	2.7
Number of fish consumers (N <sub>p</sub> ): Upper; Lower Hudson		
Twice per year	persons/y	300; 6,200
Twice per month	persons/y	90; 1,500
Twice per week	persons/y	70; 500
Ambient air total PCB concentration (Cair PCB s)	1 2	,
Dredging corridor	$ng/m^3$	23
Processing facility	$ng/m^3$	27
Individual breathing rate (BR <sub>n</sub> )		
Surrounding communities	m <sup>3</sup> /person-d	16
Project workers	m <sup>3</sup> /person-d	1.6
Cumulative exposure duration $(D_{n,s})$	/ 1	
Surrounding communities	person-d	1.347.000
Project workers	person-h	334.000
Air PCB cancer dose-response $(DR_{ADD}, rep.e)$	cases/kgpcp_intele	0.2
Air PCB noncancer dose–response $(DR_{ADD}, rep e)$	cases/kgpcp_intele	See Figure S2
Total emitted mass of primary PM <sub>e.c</sub> $(M_{\rm e})^a$	/ CFCB_IIItake	8
Heavy equipment	ka	100: 3 300
Barge traffic	kgemitted	100, 3,500
Rail transport	kgemitted	73 000
Total emitted mass of NO $(M_{\rm e})^a$	Remitted	75,000
Heavy equipment	ka	30,000, 71,000
Barge traffic	kgemitted	21 000: 30 000
Rail transport	kgemitted	2 586 000
$PM_{a-i}$ intake fraction (iE.)	Remitted	2,580,000
Primary $PM_{-2}$ heavy equipment & barge traffic	ka /ka	$5.6 \times 10^{-7}$
Primary PM <sub>2.5</sub> , heavy equipment & barge traffic	$kg_{PM_{2.5}\_intake}/kg_{i\_emitted}$	$5.0 \times 10^{-7}$
NO heavy equipment k barge traffic	$kg_{PM_{2.5}\_intake}/kg_{i\_emitted}$	$6.8 \times 10^{-8}$
NO <sub>x</sub> , neavy equipment & barge frame	$kg_{PM_{2.5}\_intake}/kg_{i\_emitted}$	$1.4 \times 10^{-7}$
$PM_{x}$ , health effect factor ( $DP_{x}$ , $\vee$ SE)	$\kappa_{SPM_{2.5}\_intake}/\kappa_{Si\_emitted}$	1.4 × 10
Above background worker <b>DM</b> <sub>2.5</sub> $\wedge$ SFPM <sub>2.5</sub> )	$DAL 1 / \kappa SPM_{2.5}$ intake	/0
Probability of fotal incident ( $\text{DE}$ ) for full time worker	μg/III unitless	1, 10 $2 \times 10^{-4}$
Number of full time equivalent workers (N $\sum_{b}^{b}$	unincess persons	2 × 10
Worker life expectancy (I E )	persons	525
worker me expectaticy (LE <sub>c</sub> )	year	43

Note: For concision, values are averaged over the considered timeframes and subpopulations of this study with exceptions below. Parameters that pertain to worker impacts are part of a separate sensitivity analysis. More detailed summaries of model parameterization are presented in the Supplemental Material.

<sup>a</sup>Estimates are presented for both low and high emission scenarios separated by semicolons. For heavy equipment (with and without workers), these estimates correspond with the Tier 4 and Tier 3 emission control standards, respectively (U.S. EPA 2004b; U.S. EPA 1998). For barge traffic, these estimates represent the range of reported emission factors between barge companies from the U.S. EPA SmartWay Carrier Performance database (U.S. EPA 2016).

<sup>b</sup>Summed across all general labor categories (c).

# Avoided Health Burden from Reduced PCBs in Fish

We estimated the health burden of PCB exposure from consumption of contaminated fish from the Site separately for Upper and Lower Hudson recreational angler subpopulations and their families, using the following equation:

$$IB_{fish,PCB} = \sum_{t=1}^{n_{years}} \sum_{p,e} \overline{C(t)}_{fish,PCB} \times \overline{IR}_{p} \times DR_{ADD_{p,fish\_PCB},e} \times SF_{PCB,e} \times N_{p}$$
(5)

where  $\overline{C(t)}_{\rm fish,PCB}$  represents a time-dependent, species- and river-section weighted average, wet-weight, fish tissue Tri + PCB concentration (kg<sub>PCB</sub>/kg<sub>fish</sub>), adjusted for cooking losses. "Tri +" refers to the sum of trichloro- through decachloro-PCB homologs.  $\overline{IR}_p$  is an annual average individual fish ingestion rate from the Site for subpopulation (p) (kg<sub>fish</sub>/person-year).

Specifically, we defined six subpopulations, three consuming fish from the Upper Hudson at annualized ingestion rates of twice per year, twice per month, and twice per week, and similarly for the Lower Hudson.  $DR_{ADD_{p,fish\_PCB,e}}$  is a dose–response factor for oral intake of PCBs (cases per kg\_{PCB\\_intake}) for health effect (e). SF<sub>PCB,e</sub> is the corresponding severity factor converting cases of effect (e) into DALYs (DALY per case). N<sub>p</sub> is the annual number of consumers.

# **Exposure** Assessment

For  $\overline{C(t)}_{fish,PCB}$ , we used model forecasts from the Record of Decision (U.S. EPA 2002b) and Responsiveness Summary (TAMS Consultants 2002). These concentrations of Tri + PCBs were forecasted by FISHRAND, a mechanistic, time-varying model that relied on solutions of differential equations and incorporated sediment and water sources predicted by fate and transport models (TAMS Consultants et al. 2000a, 2000b; TAMS Consultants and

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Input parameter	$GSD^2$	Sensitivity	% contribution
Inhalation of PCBs			
Annual average air PCB concentrations $(C_{air,PCB,s})^a$	1.1	< 0.01	< 0.01
Interspecies conversion factor <sup>b</sup>	19	< 0.01	< 0.01
Cancer dose-response factor $(DR_{ADD_{pair}, BCB, e})^{c}$	1.3	< 0.01	< 0.01
Cancer severity factor $(SF_{PCB})^d$	1.01	< 0.01	< 0.01
Noncancer dose-response factor $(DR_{ADD_{n vir BCR, e}})^{e}$	See Figure S2	< 0.01	< 0.01
Noncancer severity factor $(SF_{PCB})^f$	13.0	< 0.01	< 0.01
Inhalation of PM <sub>2.5</sub> , regional and U.S. populations			
Heavy equipment primary PM <sub>2.5</sub> emission factor <sup>g</sup>	1.3	< 0.01	< 0.01
Heavy equipment $NO_x$ emission factor <sup>g</sup>	1.3	< 0.01	< 0.01
Barge traffic primary $PM_{2.5}$ emission factor <sup>h</sup>	1.7	< 0.01	< 0.01
Barge traffic $NO_x$ emission factor <sup>h</sup>	1.4	< 0.01	< 0.01
Rail transport primary PM <sub>2.5</sub> emission factor <sup><i>i</i></sup>	1.7	0.12	0.13
Rail transport $NO_x$ emission factor <sup><i>i</i></sup>	1.7	0.87	6.70
Site-specific PM <sub>2.5</sub> intake fractions $(iF_i)^j$	4.6	0.05	0.21
Railroad PM <sub>2.5</sub> intake fractions $(iF_i)^j$	4.6	0.92	67.91
Dose–response factor $(DR_{PM_{25}})^k$	2.2	1.00	21.20
Severity factor $(SF_{PM_{25}})^k$	1.4	1.00	3.86
Inhalation of PM <sub>2.5</sub> , project workers			
Personal exposure concentration $(C_{PM_{2.5},p})^l$	3.2	< 0.01	0.01

Note: Presented values include geometric standard deviations (GSDs), sensitivity coefficients, and percent contributions to total output uncertainty. For these input parameters, we adapted an approach from MacLeod et al. (2002), assuming independent lognormal probability distributions (e.g., see Slob 1994). Following their approach, variance in each model output (cumulative health burden, DALYs) was calculated as a weighted sum of variances contributed by each input parameter with sensitivities as weights. Sensitivities were based on a 10% change in each input parameter relative to the total induced health burden in DALYs.

<sup>a</sup>Based on the average variability within dredging seasons in the site-specific ambient air PCB monitoring results used for this study (Anchor QEA and Environmental Standards, Inc. 2009; Ecology and Environment 2004, 2017).

<sup>b</sup>Accounts for uncertainty in the extrapolation of rodent data to humans as calculated by Huijbregts et al. (2005).

<sup>c</sup>Accounts for experimental uncertainty (sample size), based on the ratio of upper bound and central estimate cancer slope factors (U.S. EPA 1996).

<sup>d</sup>Based on the greatest 95th uncertainty interval for the corresponding DALY and incidence data as calculated by the Institute for Health Metrics and Evaluation (IHME 2017a). This assumes that the relative fractions of incidence for the three cancer types in these exposed populations are similar to those for the greater U.S. population (age- and sex-adjusted). Assuming these fractions are unknown would result in a maximum GSD<sup>2</sup> of 1.3 for this parameter. This would have a negligible (1%) effect on the total uncertainty in cancer health risk, since this uncertainty is driven by uncertainty in the interspecies conversion factor.

<sup>e</sup>Total uncertainty is displayed on Figure S2. Separate uncertainty distribution was applied in allometric scaling by body weight, accounting for chemical-specific interspecies differences. Interindividual variability was addressed by assuming a lognormal distribution for human variation, with an additional uncertainty distribution for the GSD of human variation. No subchronic uncertainty factor was applied, because the duration of the study by Tryphonas et al. (1991) was 55 months.

<sup>f</sup>Based on Huijbregts et al. (2005) with considerably greater uncertainty than for cancer arising from use of an average severity factor, in DALY per case, across 49 diverse, noncommunicable diseases.

<sup>g</sup>Accounts for uncertainty in the use of emission factors from Cao et al. (2016), based on variability across equipment types deemed to be most representative for this study. Furthermore, a separate uncertainty distribution was programmed in the Monte Carlo simulation to assign equal likelihood of Tiers 3 and 4 equipment.

<sup>h</sup>Reflects variability across dredging seasons based on the range of reported emission factors between 2013–2014 from the U.S. EPA SmartWay Carrier Performance database (U.S. EPA 2016). Furthermore, a separate uncertainty distribution was programmed in the Monte Carlo simulation to assign equal likelihood of each barge company in the database.

<sup>1</sup>Uncertainty distribution calculated from ranges of g per ton-mile emission factors summarized in a publication by the American Association of Railroads as provided by C. Crimmel (personal communication). Data were digitized using Plot Digitizer (version 2.6.8, Joe's Java Programs).

Uncertainty distribution based on the variability of intake fractions among models as calculated by Humbert et al. (2011).

<sup>*k*</sup>Uncertainty distribution as calculated by Gronlund et al. (2015).

Based on the variability of exposure levels reported by Lewné et al. (2007) for "construction machine operators" and "other outdoor workers exposed to diesel exhaust." Worker impacts were considered as part of a separate sensitivity analysis.

Menzie-Cura & Associates 2000). Figure S1 displays the FISHRAND forecasts for the Upper and Lower Hudson, and Table 2 displays average concentrations across the forecast periods. Reductions in average concentration for implementing SC over NA were 49% and 39% for the Upper and Lower Hudson, respectively. Similarly, reductions associated with implementing SC&ED over SC, were 36% and 16% for the Upper and Lower Hudson, respectively.

Using 6-y average PCB concentrations, we estimated the cumulative health burden from 2004, the model's assumed start date of the remediation, to the furthest available forecast: 2067 for the Upper Hudson and 2046 for the Lower Hudson. Six-year averaging was based on the estimated exposure duration for an average adult angler, consistent with the prior human health risk assessment for the Site. We also applied the same central tendency PCB cooking loss factor of 20% used in that prior risk assessment (TAMS Consultants and Gradient Corporation 2000).

In our uncertainty analyses, we considered additional FISHRAND model forecasts. Because the central estimate forecasts under NA and SC are highly uncertain and may overestimate the rate of decline of PCBs, we derived geometric standard deviations (GSDs) for  $\overline{C(t)}_{fish,PCB}$ , based on the ratios of estimated upper bound and central estimate forecasts (Table

S4). Similarly, for the SC&ED scenario, we derived GSDs based on the forecasts under the "REM (6-y 2.5% resuspension)" scenario as an equivalent estimated upper bound.

To evaluate population-level fish consumption from the Site, we used data from several site-specific surveys. We first used data on fishing effort from the 2007 New York Statewide Angler Survey [provided by N. Connelly (personal communication); Connelly and Brown 2009], specific to counties surrounding the Site. In particular, we estimated the annual average number of anglers who fish from the Site, both for the Upper and Lower Hudson. We then coupled these estimates with original data on consumption habits of anglers and their families from two surveys administered by the New York State Department of Health (U.S. EPA 2017). These calculations accounted for the proportions of anglers who reported consuming fish from the Site, and those who also reported sharing fish with their families, at different annualized ingestion rates (twice per year, twice per month, and twice per week). We assumed an average household size of three persons (U.S. Census Bureau 2017). Table S5 summarizes these results. To address limitations of these surveys, we corrected estimates of Np, such that corresponding estimates of total populationlevel consumption (kg<sub>fish</sub>/y) matched those calculated using data from two more comprehensive, site-specific creel surveys

(Normandeau Associates 2003, 2007). Table S6 summarizes these population-level estimates. Furthermore, we characterized the uncertainty of  $\overline{IR}_p$  assuming uniform distributions ranging from one to three meal-per-week equivalents. To convert meals into mass equivalents, we assumed a serving size of 0.5 lb(227 g)/meal, consistent with the prior human health risk assessment (TAMS Consultants and Gradient Corporation 2000). Tables S7 and S8 further summarize these input data.

#### Dose–Response and Severity

The term  $DR_{ADD_{p,fish\_PCB,e}} \times SF_{PCB,e}$  in Equation 5 includes two general health effects: cancer and noncancer. In both cases,  $DR_{ADD_{p,fish\_PCB,e}}$  is a function of average daily dose (ADD),  $ADD_{p,PCB} = \overline{C(t)}_{fish,PCB} \times \overline{IR}_{p}/(BW \times \Delta t)$ , with body weight BW (kg) and  $\Delta t = 365 \text{ d/y}$ .

We derived the cancer  $DR_{ADD_{p,fish\_PCB,e}}$  from the central estimate "High Risk and Persistence" Cancer Slope Factor for PCBs from the Integrated Risk Information System (IRIS) (U.S. EPA 1996). This estimate was consistent with the prior human health risk assessment (TAMS Consultants and Gradient Corporation 2000). We converted the slope factor into a human-equivalent lifetime dose, the inverse of which yielded a linear  $DR_{ADD_{p,fish\_PCB,e}}$ accounting for PCB carcinogenicity. We considered three cancer types based on conclusions of the International Agency for Research on Cancer (IARC 2016). These types included malignant melanoma, non-Hodgkin's lymphoma, and breast cancer. Then, using data from the 2015 Global Burden of Disease Study for these cancer types (IHME 2017a), we calculated an incidence-weighted average SF<sub>PCB,e</sub> for the U.S. population, adjusted for age and sex.

For noncancer, we adapted recent work by the World Health Organization's International Programme on Chemical Safety to probabilistically incorporate a nonlinear dose–response relationship (Chiu et al. 2018; Chiu and Slob 2015; WHO 2014). This approach enabled us to predict the human population dose–response relationship based on experimental animal data, incorporating a probabilistic characterization of uncertainty in extrapolating from animals to humans and in the degree of variability in the human population.

We based our prediction on the critical study by Tryphonas et al. (1991) that forms the basis of IRIS's current reference dose for Aroclor 1254 (U.S. EPA 1994). The U.S. Environmental Protection Agency (U.S. EPA) determined that Aroclor 1254 (  $\sim 54\%$  chlorine by weight) was the commercial PCB mixture that most resembled the homolog distribution in Hudson River fish (TAMS Consultants and Gradient Corporation 2000). Using data from Tryphonas et al. (1991) (summarized in Table S9), we conducted benchmark dose modeling of decreases in immunoglobulin M. This is a sensitive end point, selected based on high statistical significance and relatively large effects reported in the study. We used the web portal benchmarkdose.org (Shao and Shapiro 2018), setting the benchmark response at 50%, a value similar to the lowest observed adverse effect-level for multiple effects reported in the study (Tryphonas et al. 1991). To account for model uncertainty, we used Bayesian model averaging (three Markov Chains for eight different models) as described by Shao and Shapiro (2018). The remaining probabilistic extrapolations were conducted following approaches described by Chiu and Slob (2015) and Chiu et al. (2018).

Figure 1 displays the nonlinear dose–response relationship for noncancer PCB effects, the slope of which is equal to  $DR_{ADD_{p,fish,PCB,e}}$  (Figure S2). Also shown are 95% confidence intervals (CI) of average daily doses (mg/kg-d), which are functions of  $C(t)_{fish,PCB}$  and  $\overline{IR}_p$ , under the No Action scenario (2004–2009), for the three Upper Hudson subpopulations. These subpopulations were predicted to have had the highest exposures because they were consuming fish from the Upper Hudson, for which average predicted PCB concentrations were about a factor of two higher than those for the Lower Hudson (Table 2). Moreover, 2004–2009 is the time frame when concentrations were highest among the forecast periods (Figure S1). High values of mean estimates at very low doses were unstable due to their being driven by extreme values from the Monte Carlo analysis; the



Subpopulation | Twice/yr | Twice/mo | Twice/wk

Figure 1. Noncancer PCB dose–response relationship corresponding to a 50% decrease in immunoglobulin M. Curved (black) solid line = median. Curved (black) dashed line = arithmetic mean. Surrounding (dark gray) area = 95% confidence interval. Vertical (colored) dashed lines = 95% confidence intervals of average daily doses (mg/kg-d) for three subpopulations: Upper Hudson anglers and their family members consuming fish at frequencies of *a*) twice per year, *b*) twice per month, and *c*) twice per week during the 2004–2009 timeframe.

median was therefore used as the measure of central tendency. Median estimates of noncancer  $DR_{ADD_{p,fish\_PCB,e}}$  ranged several orders of magnitude between the twice-per-year  $(1 \times 10^{-4} cases / kg_{PCB\_intake})$  and twice-per-week (690 cases / kg\_{PCB\\_intake}) consumer subpopulations. In comparison, the median cancer  $DR_{ADD_{p,fish\_PCB,e}}$  was 0.6 cases / kg\_{PCB\\_intake} for all subpopulations, assuming linearity (Table 2). Thus, noncancer risk could greatly surpass that of cancer risk for highly exposed individuals.

Because the specific noncancer end points anticipated are not as clear as they are for cancer (Faroon and Olson 2000; Faroon and Ruiz 2016), for the noncancer  $SF_{PCB,e}$  we used an incidenceweighted average of 2.7 DALYs per case, based on the work of Huijbregts et al. (2005). This incidence-weighted average accounts for 49 diverse, noncommunicable diseases.

#### Health Burden of Increased Air Emissions of PCBs

We estimated the induced burden of increased air emissions of PCBs on surrounding-community subpopulations (p) across all dredging seasons (s) using the following equation:

$$IB_{air,PCB,p} = \sum_{s} C_{air,PCB,s} \times BR_{p} \times D_{p,s} \times DR_{ADD_{p,air\_PCB},e} \times SF_{PCB,e}$$
(6)

where  $C_{air,PCB,s}$  represents an above-baseline, dredging-season average, ambient air concentration of total PCBs ( $kg_{PCB}/m^3$ ); BR<sub>p</sub> is the average individual breathing rate ( $m^3/d$ ); D<sub>p,s</sub> is a spatially differentiated, cumulative exposure duration (person-d). The dose–response (DR<sub>ADD<sub>p,air\_PCB,e</sub>) and severity factor (SF<sub>PCB,e</sub>) have the same interpretations as in Equation 5.</sub>

#### **Exposure** Assessment

Cair.PCB.s is based on ambient air data from a comprehensive monitoring program (Anchor QEA and Environmental Standards, Inc. 2009; Ecology and Environment 2004, 2017). Briefly, General Electric employed 24-h samplers that continuously monitored airborne PCBs at the processing facility and along the dredging corridor. We stratified these data by dredging season and location (dredging corridor and processing facility), after excluding background data and treating nondetects as one-half the method detection limit. We then applied arithmetic mean total PCB concentrations for each stratum as Cair, PCB, s. To estimate the health burden on surrounding communities induced by the remediation (i.e., above the baseline risk), we used air PCB data from the Predredging, Background Monitoring Results (2005-2006) reported by Ecology and Environment (2017). For this calculation, we set  $BR_{communities}$  to 0.66 m<sup>3</sup>/h or 16 m<sup>3</sup>/d, an average long-term daily inhalation rate for the general population (U.S. EPA 2011).

To derive  $D_{p,s}$  for the surrounding communities, we used census-based population count data (CIESIN and Columbia University 2016) and a geographic information system of the Site [provided by M. Cheplowitz (personal communication)]. Specifically, we estimation the number of individuals residing within 500 m of the dredging for each dredging season. This was the distance from the Site at which the median ambient air PCB concentration reached a level near background (Figure S3). Population count estimates differed for each dredging season (Table S10) because dredging progressed in a general north-tosouth fashion (U.S. EPA 2017), thus affecting different community subpopulations.

#### Dose–Response and Severity

We evaluated cancer-related health impacts associated with PCB inhalation using a  $DR_{ADD_{n,air,PCB},e}$  derived from the central estimate

"Low Risk and Persistence" Cancer Slope Factor for PCBs (U.S. EPA 1996). For noncancer, we assumed the same nonlinear dose–response relationship for inhalation exposures as the dose–response relationship for ingestion, due to a lack of dose–response data for this exposure pathway.  $SF_{PCB,e}$  is also the same as that for fish consumption.

#### Health Burden of Primary and Secondary PM<sub>2.5</sub> Emissions

Although diesel exhaust is a complex mixture of organic and inorganic chemicals in gas and particulate phases (Harbison et al. 2015), we focused our assessment on the impacts of primary and secondary  $PM_{2.5}$  for three primary reasons: *a*) reducing ambient air concentrations of  $PM_{2.5}$  has been a fundamental aim of national emission-control legislation for nonroad vehicles in the United States (U.S. EPA 2004b); *b*) the particulate fraction of diesel exhaust has been considered to be the "risk driver" of its health effects (Hesterberg et al. 2011); and *c*) available epidemiological data for ambient  $PM_{2.5}$  are much more reliable for quantitative risk assessment than those for the preferred surrogate of exposure to diesel exhaust, elemental carbon (Möhner and Wendt 2017; Morfeld and Spallek 2015; Pronk et al. 2009).

For the regional and U.S. populations, we used the following equation for all  $PM_{2.5}$  precursors (i) and emission source categories (j):

$$IB_{PM_{2.5},general population} = \sum_{i,j} M_{i,j} \times iF_i \times DR_{PM_{2.5}} \times SF_{PM_{2.5}}$$
(7)

where  $M_{i,j}$  represents the total emitted precursor mass ( $kg_{i\_emitted}$ ); iF<sub>i</sub> is the spatially differentiated PM<sub>2.5</sub> intake fraction for each considered precursor ( $kg_{PM_{2.5\_intake}}/kg_{i\_emitted}$ ); DR<sub>PM<sub>2.5</sub></sub> is a dose– response factor for PM<sub>2.5</sub> (deaths per  $kg_{PM_{2.5\_intake}}$ ); and SF<sub>PM<sub>2.5</sub> is the corresponding severity factor (DALYs per death).</sub>

#### **Exposure** Assessment

We derived  $M_{i,j}$  for primary PM<sub>2.5</sub> and NO<sub>x</sub> (i) using an emission-factor based approach for three source categories (j): nonroad diesel-powered heavy equipment, barge traffic, and rail transport (line-haul operation). Tables S11-S13 summarize the input parameterization. For heavy equipment, we obtained data on the number and types of equipment from an estimated project inventory of primary diesel-powered, nonroad heavy equipment [provided by M. Cheplowitz (personal communication)] and data on equipment specifications from manufacturers and distributors. For each equipment type, we then estimated the total number of hours of effective operation and idling from data in Weekly Productivity Summaries. For similar equipment types, we applied in-use load factors, a measure of how hard an engine is working, and emission factors from a recent study (Cao et al. 2016). Because emission control technology can substantially reduce emissions (Clark et al. 2002; Khalek et al. 2009, 2013), and because the project began around the time when the U.S. EPA phased in the latest emission control standard, Tier 4 (U.S. EPA 2004b), we probabilistically assessed emissions and impacts for Tier 3 (U.S. EPA 1998) and Tier 4 heavy-equipment scenarios, assuming equal likelihoods.

Unlike for heavy equipment, we did not have detailed data on equipment characteristics and operating times for barge traffic. Accordingly, we applied emission factors for this source category in units of mass of emitted pollutant per (short) ton-mile of transported sediment and water (g per ton-mile). We obtained riverbarge emission factors from the U.S. EPA SmartWay Carrier Performance database (U.S. EPA 2016). For the 92,590 reported total barge miles traveled (Louis Berger Group et al. 2017), we assumed equal distances to and from the processing facility. For trips to the facility, we calculated an average mass of transported load per barge based on the reported tonnage of material shipped off-site, the total volume of water treated and returned to the river, and the total number of unloaded barges for each dredging season. To account for unloaded barge returns, we scaled the emission factors proportional to an estimated weight reduction. For this, we obtained gross tonnage data from a local distributor (Sterling Equipment, Inc.) for equipment that reasonably matched project photographs and specifications in the Phase 1 Evaluation Report (Louis Berger Group 2010) and Remedial Action Work Plans.

For rail transport, we applied weighted-average emission factors (g per ton-mile) representing U.S. Class 1 railroad companies (2011-2015). These data were also from the EPA SmartWay Carrier Performance database (U.S. EPA 2016). We calculated the mass of sediment delivered to each of seven reported hazardous waste landfills, using project manifest data for all remediation seasons and reported trips. We then estimated the distance traveled to and from each landfill as the shortest path, using the Network Analyst tool in Esri ArcMap (version 10.5) on the U.S. Railroad Lines data set (U.S. Department of Transportation 2017). This estimation resulted in seven distinct railroad routes. We accounted for each unloaded return as we did for barge traffic, using project data on tare weights of empty project gondolas, the average number of locomotives per U.S. unit train for years 2009-2015 (Association of American Railroads 2017), and assuming a locomotive weight of 423,500 lb [received from C. Crimmel (personal communication); Norfolk Southern Company 2014].

To link emissions of  $PM_{2.5}$  and  $NO_x$  with corresponding intakes of primary and secondary  $PM_{2.5}$  by the regional and U.S. populations, we derived spatially differentiated estimates of  $iF_i$ for both precursors.  $iF_i$  represents the incremental  $PM_{2.5}$  mass intake, summed over all exposed individuals over time, per unit mass of emitted precursor ( $kg_{PM_{2.5\_intake}}/kg_{i\_emitted}$ ) (Bennett et al. 2002). For the Site and for each of the seven routes, we derived estimates of  $iF_i$  (Table 2 and Table S13) by coupling 2015 population density data for North America (CIESIN and Columbia University 2016) with marginal increases in ambient  $PM_{2.5}$  concentrations associated with ground-level emissions. These concentrations were simulated by a mechanistic air pollution model for the greater region of North America, known as Intervention Model for Air Pollution version 1.2.0 (Tessum et al. 2017). We assumed the same average breathing rate of  $16 \text{ m}^3/\text{d}$  as for the PCB-inhalation exposure pathway (U.S. EPA 2011).

#### Dose-Response and Severity

To quantify the health burden associated with population intakes of primary and secondary  $PM_{2.5}$ , we applied a combined  $DR_{PM_{2.5}}$  and  $SF_{PM_{2.5}}$  of 78 DALY/kg $_{PM_{2.5}$ \_intake from Gronlund et al. (2015), accounting for cardiopulmonary and lung-cancer mortality.

#### Sensitivity Analysis: Health Risks to Project Workers

To assess the potential health burden of ED on project workers, we considered two worker subpopulations to account for their estimated differences in exposure. These subpopulations included processing facility workers and dredging corridor workers.

#### Health Burden of Inhalation of PCBs and PM<sub>2.5</sub>

The general framework for estimating occupational health risks from PCB inhalation is the same as for surrounding communities (Equation 6). For this estimation, we set  $BR_{workers}$  to  $1.6 \text{ m}^3/\text{h}$ , an average hourly inhalation rate for outdoor workers (U.S. EPA 2011). For facility workers, we derived  $D_{p,s}$  from projections in

Remedial Action Work Plans and from operation dates reported in Weekly Productivity Summaries. For dredging corridor workers, we applied a similar method using the estimated project inventory supplied by M. Cheplowitz mentioned previously. Tables S10 and S14 summarize these estimates.

To quantify occupational health risks due to  $PM_{2.5}$  inhalation from the use of diesel-powered heavy equipment, we used the following equation for all worker subpopulations (p) and dredging seasons (s):

$$IB_{PM_{2.5},workers} = \sum_{p,s} C_{PM_{2.5},p} \times BR_p \times D_{p,s} \times DR_{PM_{2.5}} \times SF_{PM_{2.5}}$$
(8)

where  $C_{PM_{2.5},p}$  represents an above-background, personal exposure concentration of diesel  $PM_{2.5}$  during a work shift  $(kg/m^3)$ ; individual breathing rate  $(BR_p)$  and cumulative exposure duration  $(D_{p,s})$  are the same as for the PCB-inhalation exposure pathway;  $DR_{PM_{2.5}}$  and  $SF_{PM_{2.5}}$  have the same meanings as for the regional and U.S. populations.

We obtained estimates of  $C_{PM_{2,5},p}$  from Lewné et al. (2007) for *a*) operators of nonroad heavy equipment and *b*) support workers frequently in close proximity to heavy equipment. As previously described, we probabilistically assessed emissions and impacts for Tier 3 and Tier 4 heavy equipment scenarios, assuming equal likelihoods. To assess the increased risk attributable to the remediation, we subtracted from each  $C_{PM_{2,5},p}$  an estimated regional background concentration of  $8.3 \,\mu\text{g/m}^3$  for Albany/ Schenectady, New York (Shaddick et al. 2018).

#### Health Burden of Fatal Occupational Incidents

To estimate the health burden of fatal occupational incidents, we used the following equation for all general labor categories (c) and dredging seasons (s):

$$IB_{fatal incidents, workers} = \sum_{c, s} PF_c \times N_{c,s} \times LE_c$$
(9)

where  $PF_c$  represents the probability of a fatal occupational incident for one full-time equivalent worker;  $N_{c,s}$  is the number of full-time equivalent workers;  $LE_c$  is the average life expectancy (y), a function of age. Under this framework,  $\sum_{c,s} PF_c \times N_{c,s}$  estimates the number of fatal incidents, and  $LE_c$  quantifies the DALYs if a fatal incident were to occur. To propagate uncertainty, we modeled  $\sum_{c,s} PF_c \times N_{c,s}$  as a combination of assumed-

independent random binomial variables with each distribution defined as  $B(N_{c,s}, PF_c)$ .

To derive  $PF_c$ , we adapted an approach used by the U.S. Bureau of Labor Statistics (U.S. BLS) for their Census of Fatal Occupational Injuries. Each year, U.S. BLS publishes national fatal injury rates using the Standard Occupational Classification (SOC) system (U.S. BLS 1997). We matched project general labor categories to detailed SOC occupations based on work descriptions in Remedial Action Work Plans. We then used data on fatal occupational injury counts and the number of at-work employees for years 2011–2015, as provided by J. Kang (personal communication), calculating for each general labor category an average fatal injury rate (PF<sub>c</sub>) for one full-time equivalent worker (2,000 h/y).

To estimate  $N_{c,s}$ , we used projections from the Remedial Action Work Plans, converting these to a full-time equivalent basis. We then estimated  $LE_c$  based on median ages of at-work employees in matched SOC categories as provided by J. Kang (personal communication) using the Global Burden of Disease

2015 Reference Life Table (IHME 2017b). Table S2 presents estimates of  $PF_c$ ,  $N_{c,s}$  and  $LE_c$  for each general labor category and the corresponding SOC categories.

# Results

# Health Burdens from PCBs in Fish

Figure 2 compares the central tendency, baseline (NA) health burden from consumption of Hudson River fish and the central tendency health burdens under the SC and SC&ED scenarios. This comparison highlights the relative contributions of cancer versus noncancer risk, and Upper versus Lower Hudson anglers and their families, to the total burden. A burden of 112 DALYs (90% CI: 2, 7,676) was estimated using Equation 5 under NA for the considered time frames (2004-2067 for the Upper Hudson and 2004-2046 for the Lower Hudson). The burden under SC was estimated at 59 DALYs (90% CI: 1, 4,104), approximately half of the baseline burden. Adding dredging (SC&ED) further reduced the burden to 36 DALYs (90% CI: 1, 2,618). For all three scenarios, noncancer risk for Upper Hudson anglers and family members comprised most of the total burden (62%-75%), due to a relatively small subpopulation of consumers with frequent consumption (twice per week). The rest of the total burden was attributed to noncancer risk for Lower Hudson anglers and family (15%-21%), cancer risk for Lower Hudson anglers and family (7%-14%), and cancer risk for Upper Hudson anglers and family (2%-3%).

The first bar in Figure 3 shows the avoided burden due to ED (i.e., SC&ED over SC), including the 90% CI. As shown, ED resulted in 15 avoided DALYs (90% CI: 0, 1,376) of health benefit due to long-term reductions in fish tissue PCB concentrations.

#### Induced Health Burdens from Environmental Dredging

Figure 3 summarizes estimates of induced burdens from ED on the surrounding communities and regional and U.S. populations associated with increased air emissions of PCBs and primary and secondary PM<sub>2.5</sub>. Results for each impact are described later in this paper.

# Inhalation of PCBs

Using Equation 6 with parameters summarized in Table 2 yielded an induced burden of  $2 \times 10^{-3}$  DALYs (90% CI:  $1 \times 10^{-4}$ ,  $2 \times 10^{-2}$ )

for the surrounding communities attributed to increased PCB emissions to air during the dredging (Figure 3). From Table 2, Table S10, and Figure S3, it is evident that annual averages of total airborne PCB concentrations measured during the remediation were well below the U.S. EPA's level of concern of  $110 \text{ ng/m}^3$ . This finding suggests the applied best management practices were successful overall in limiting increases in air PCB concentrations. This finding was also noted during the second Five Year Review for the Site (Ecology and Environment 2017). For the surrounding communities, estimates of inhalation exposures are likely conservative, given that the underlying data represent nearest-receptor locations. In any case, the burden induced by increased air emissions of PCBs during the ED operation appears to have been minor in comparison with other induced burdens and to the potential health benefits of long-term reductions in fish tissue PCB concentrations.

# Inhalation of PM<sub>2.5</sub>

As summarized in Table 2 (with more detailed results in Table S11), total emissions of primary  $PM_{2.5}$  and  $NO_x$  from project heavy equipment under the Tier 3 scenario amounted to 3,300 kg and 71,000 kg, respectively. These yielded, in combination with values from Table 2, a total PM2.5 intake of 0.007 kg across the regional and U.S. populations. Relative contributions of primary PM<sub>2.5</sub> and NO<sub>x</sub> to this total were 28% and 72%, respectively. When assuming better emission-control technology under the Tier 4 standard, emissions of PM2.5 and NOx from heavy equipment were considerably reduced to 100 kg and 30,000 kg, respectively. This assumption led to a PM2.5 intake of 0.002 kg across the regional and U.S. populations, with a much lower relative contribution of 3% from primary PM<sub>2.5</sub> vs. 97% from NO<sub>x</sub>. Assuming equal likelihood for both heavy equipment scenarios resulted in a median health burden of 0.3 DALYs (90% CI: 0.05, 2) for these populations. Barge traffic emissions (Table 2 and Table S12) resulted in an additional cumulative  $PM_{2.5}$  intake of 0.002 kg and a health burden of 0.1 DALYs (90% CI: 0.03, 1).

Site-specific emissions from project heavy equipment and barge traffic were limited in comparison with emissions from rail transport of sediment across the United States. Assuming project trains used the shortest path to and from each landfill, we



**Figure 2.** Comparison of median cumulative induced health burden (DALYs) associated with bioaccumulation of Tri + PCBs in Hudson River fish and exposure through fish consumption. Estimates of induced health burden are presented for the three scenarios: *a*) No Action (IB<sub>fish,PCB,NA</sub>), *b*) Source Control (IB<sub>fish,PCB,SC</sub>), and *c*) the selected remedy combining SC and ED (IB<sub>fish,PCB,SC&ED</sub>). Results are stratified by health outcome (cancer vs. noncancer) and river section (Upper Hudson vs. Lower Hudson).



Figure 3. Comparison of avoided and induced health burdens of environmental dredging (ED) for the Hudson River PCBs Superfund Site. The thinner (red) bars reflect the fifth and 95th percentiles of each Monte Carlo realization. All central estimates are medians, except for fatal occupational incidents, which is based on the arithmetic mean. Worker impacts are included as a separate sensitivity analysis, in case they can be attributed to ED.

estimated that they traveled a total of 1,021,255 railroad miles to ship the reported 3,165,820 tons of sediment (Table S13). As shown in Table 2, this estimation corresponded to 73,000 kg of primary  $PM_{2.5}$  and 2,586,000 kg of  $NO_x$  emitted by project locomotives in total. These emissions resulted in a substantial cumulative population  $PM_{2.5}$  intake of 0.410 kg, over 60 times higher than the total intake from all other sources. The corresponding health burden was 32 DALYs (90% CI: 7, 145).

The nearest landfill used for the project was CWM Chemical Services in Model City, New York. This landfill was an estimated 334 railroad miles from the Site, yet only two project railcars were shipped to this destination out of an estimated 30,000, while approximately 70% of project unit trains delivered waste to landfills over 1,500 railroad miles from the Site (Table S13). If all project trains delivered to the nearest landfill, the corresponding health burden would have been about a factor of 5 lower [7 DALYs (90% CI: 1, 32)].

#### Net Health Benefits of Environmental Dredging

Figure 4 summarizes all estimated adverse health impacts and health benefits of ED, including uncertainty evaluated via Monte Carlo simulation. As previously mentioned, ED achieved an estimated avoided burden of 15 DALYs (90% CI: 0, 1,376) (Figure 4A). The large right skew in the distribution of total avoided burden reflects the large uncertainty associated with the noncancer dose–response for PCBs. On the other hand, potential health burdens induced by ED on surrounding communities and the regional and U.S. populations amounted to 33 DALYs (90% CI: 8, 146) (Figure 4B). As shown in Figure 3, most of these adverse impacts were attributed to PM<sub>2.5</sub> from rail transport [32 DALYs (90% CI: 7, 145)].

Figure 4C shows the net health benefit of ED [i.e., the distribution of the difference between the avoided burden (benefit) and the induced burden (risk)]. The median of this distribution was -11 DALYs of benefit (90% CI: -138, +1,326) or 11 DALYs of induced burden. Although the upper confidence bounds suggest the possibility of larger benefits, only 39% of these Monte Carlo samples corresponded to a positive net health benefit for the ED operation.

#### Sensitivity Analysis: Health Burdens for Workers

The net health benefit of the ED operation would be further diminished if it posed increased risks to project workers. Equations 6 and 8 with parameter values summarized in Table 2 yielded induced burdens of  $1 \times 10^{-4}$  DALYs (90% CI:  $9 \times 10^{-6}$ ,  $1 \times 10^{-3}$ ) and 0.4 DALYs (90% CI: 0.03, 5) on workers due to inhalation of PCBs and PM<sub>2.5</sub>, respectively. As shown in Figure 3, these burdens were of similar orders of magnitude as their general-population counterparts, though they may be reduced if effective personal protective equipment were used. Worker impacts from barges and rail transport were not assessed, due to a lack of available exposure data.

According to Equation 9, the health burden of fatal occupational incidents was 14 DALYs (90% CI: 0, 46), which is considerably higher than the other considered worker impacts. Because the estimated median life expectancy of this workforce is 47 y (Table S2; IHME 2017b), this central tendency health burden corresponds approximately to a 30% chance  $(14/47 \times 100\%)$  of a fatal occupational incident occurring for this project. Including workers in the overall assessment would result in a net health benefit of -20 DALYs (90% CI: -163, +1,312) or 20 DALYs of induced burden for the ED operation (Figure S4).



Figure 4. Stochastic health benefit–risk comparison for the Hudson River PCBs Superfund Site environmental dredging (ED) remediation. Results were generated via Monte Carlo simulations accounting for parameter variability and uncertainty. A) Avoided Health Burden  $(AB_{ED})$  = induced health burden of fish consumption on surrounding communities under Source Control (SC) – induced health burden of fish consumption on surrounding communities under SC&ED; B) Induced Health Burden ( $IB_{ED}$ ) = total health burden of ED on the regional and U.S. populations (including surrounding communities) from increased air emissions of PCBs, and primary and secondary PM<sub>2.5</sub>; C) Net Avoided Health Burden (Net health benefit<sub>ED</sub>) =  $AB_{ED} - IB_{ED}$ . Dotted or dashed vertical lines correspond to the fifth, 10th, 25th, 50th, 75th, and 90th percentiles when read from left to right. The solid (red) vertical line through zero denotes a net of 0 avoided DALYs (i.e., benefits = risks). Values to the left of this line represent net risks while values to the right of this line represent net benefits.

### Discussion

#### Strengths and Limitations

In this study, we conducted a comparative human health risk assessment to evaluate trade-offs in health benefits and risks

accompanying one of the largest remediation projects in U.S. history. Use of DALYs as a common metric facilitated extensive accounting of population-level effects beyond local communities. Other strengths of our approach include: a) use of site-specific data in all parts the analysis; b) incorporation of noncancer health benefits from reduced exposure; and c) detailed application of uncertainty analysis.

Considering both avoided and induced health burdens in our analysis suggests that the dredging performed at the Hudson River PCB Superfund Site might not have led to an overall net positive human health impact in excess of source control. Health risks primarily resulting from long-distance transport of sediment appear to negate the benefits of dredging. For the considered impacts, the results suggest there is <50% likelihood that the long-term population health benefits of the dredging remedy would exceed health risks to surrounding communities and the regional and U.S. populations (median in Figure 4C).

The difference between this study's and the U.S. EPA's results at the time of remedy selection (U.S. EPA 2002a) relates primarily to differences in the scope of the health risk assessments. Both approaches, however, have the potential to inform future decision-making in a complementary way.

- First, the U.S. EPA's assessment focused on individual health risk to community members, whereas this study assessed long-term, *population* health risks to communities and the regional and U.S. populations. Our assessment included risks due to PM<sub>2.5</sub> precursor emissions from longdistance transport of sediment. The individual perspective is useful for identifying and protecting individuals at high risk. Our analysis of survey data from the New York State Department of Health suggested that a small percentage of Upper Hudson anglers may be at high risk of noncancer health effects from frequent fish consumption. In the absence of active remediation, it would thus be imperative to maintain institutional controls that target these individuals. The population perspective, on the other hand, is useful for comparing potential benefits and risks associated with such a large remediation project.
- Second, in contrast to the U.S. EPA's assessment, this study took a cumulative temporal perspective rather than focusing on the predicted years at which fish tissue PCB concentrations would reach levels deemed acceptable for individuals. Like the contrast between individual-level vs. populationlevel risk, both perspectives may be informative in future decision-making.

This case study also has several important limitations, mostly related to large uncertainties in both the data and analysis. One of the largest uncertainties was in estimating noncancer health benefits of long-term reductions in fish tissue PCB concentrations, primarily due to uncertainties in the noncancer dose–response relationship spanning more than three orders of magnitude. This uncertainty is driven largely by the use of experimental animal data in deriving the predicted human population dose–response. IRIS is currently reassessing the noncancer human health hazards of PCBs and corresponding dose–response information. It is possible that these uncertainties can be reduced based on updated toxicity data and dose-response analyses.

Another significant uncertainty in estimating health benefits of ED was in quantifying PCB exposures from fish consumption. For instance, the second Five Year Review discusses several important differences between the FISHRAND model assumptions and the remediation as implemented (Louis Berger Group et al. 2017). In addition, our approach for quantifying fish consumption was limited to combining data from several data sources, deemed to be the best available, each with their own limitations (Tables S7 and S8). Nonetheless, our estimates of limited consumption from the Upper Hudson are generally consistent with the only measurement-based assessment of PCB exposures from fish consumption conducted for the Site. Specifically, Fitzgerald et al. (2007) reported no significant differences in measured total serum PCB concentrations between older residents of Upper Hudson communities and an upstream control population. Both populations had low rates of reported fish consumption and PCB body burdens similar to populations with no unusual exposures (Fitzgerald et al. 2007).

Total uncertainty in the induced health burden of ED was driven by only two parameters: the intake fractions for  $PM_{2.5}$  (mostly, for secondary  $PM_{2.5}$  from  $NO_x$ ) used to estimate impacts from train emissions (68%), and the corresponding dose–response factor (21%) (Table 3). Nonetheless, there were several sources of uncertainty beyond our means to quantitatively evaluate. With respect to  $PM_{2.5}$ , we used a linear dose–response factor because the nonlinear dose–response relationships are very uncertain in regions with relatively low background concentrations.

Our assessment also relied on national statistics to assess the health burden of fatal occupational incidents. The specific jobs for this remediation project may have been more or less hazardous than the more general categories to which we matched (Leigh 2011). These limitations, coupled with the possibility of similar risks occurring for workers in the absence of dredging, prompted us to first conduct the assessment without considering potential occupational health risks and then consider them separately in a sensitivity analysis. Our estimate of a 30% chance of a fatal occupational incident is consistent with the actual occurrence of one during the remediation. Specifically, a 39-y-old worker who had been involved in a support activity aimed at protecting cultural resources drowned after his boat went over a dam (Nelson 2009). This occurrence underscores the importance of rigorous occupational health and safety programs in future projects with careful attention to physical hazards.

Several other types of impacts were not included in our analysis. First, we did not address the trade-off between health risks associated with PCB exposure and health benefits of nutrients, such as the longer-chain omega-3 fatty acids, docosahexaenoic acid and eicosapentaenoic acid, which also bioaccumulate in fatty tissues. PCBs and omega-3 fatty acids may have counteractive effects on similar end points, such as those pertaining to early cognitive development and cardiovascular health (Turyk et al. 2012). Regarding other potential exposure pathways, previous work determined that baseline risks from incidental ingestion of sediment and water, dermal contact with sediment and water, and inhalation of volatilized PCBs were generally below levels of concern (TAMS Consultants and Gradient Corporation 2000). In future work, it may be worth evaluating potential health risks associated with consumption of resident waterfowl. This exposure pathway is complicated, however, by an inability to readily distinguish between resident and migratory waterfowl of the same species (HRNRT 2013). There is also an ongoing investigation of PCB contamination along the Hudson River floodplain (U.S. EPA 2017), which was not evaluated in this study. Additionally, we were unable to assess potential health risks of the source control action, and we did not evaluate the general benefit of full use of the Site without institutional controls. Finally, because this study focused on human health risk, potential ecological, social, and economic effects of environmental dredging were not addressed.

#### Implications for Decision-Making and Future Research

The results of this study underscore the importance of considering both potentially avoided and induced risks of remediation alternatives. Moreover, they suggest that ED is accompanied by important trade-offs pertaining to human health. These trade-offs become evident only when the scope of analysis is expanded beyond local populations. Doing so in our assessment has highlighted a need to understand and minimize adverse impacts of remediation projects, which is in line with the U.S EPA's green remediation efforts (U.S. EPA 2010).

An important issue that became apparent in our analysis was the induced health burden of ED on the regional and U.S. populations, especially people residing near railroads. These risks were driven by long-distance transport of sediment and associated emissions of primary PM2.5 and NOx. Delivering sediment to landfills up to 2,500 miles (and possibly farther) away from the Site may have induced health risks that match and potentially exceed the health benefits of ED. Assuming a fuel-consumption factor of 0.002 gal/ton-mile would suggest that the ED operation consumed approximately 12,100,000 gallons of diesel fuel from rail transport alone (BTS 2012). Thus, future remediation projects should pay more attention to reducing transport distances and transported quantities. Furthermore, as the existing U.S. locomotive fleet is eventually phased out and replaced by locomotives compliant with the Tier 4 emission control standard, air emissions and associated health risks from rail transport in general are expected to decrease appreciably (U.S. EPA 2008), stressing the importance of such standards. Expanding the scope of analysis, however, to include broader population impacts suggests that future decision-making may need to consider a wider range of health impacts.

More broadly, the results of this study raise the question of how remediation can be conducted in a way that best maximizes health benefits while minimizing health risks? In the case of the Hudson River, the selected remedy may have satisfied Superfund's risk reduction objectives based on PCBs, but the negative populationlevel health impacts of, for example, combusting over 12 million gallons of diesel fuel were not directly considered in U.S. EPA's decision-making or design. Based on our analysis, health risks attributed primarily to the generation of PM2.5 during longdistance transport of sediment by train may have outweighed health benefits from reductions in fish tissue PCB concentrations. Our analysis also demonstrates that PM<sub>2.5</sub>-related risks could have been markedly reduced if dredged materials were disposed of locally instead of being processed and shipped via rail across the country. The research conducted herein clearly demonstrates the need to consider such impacts and provides an impetus to reduce them to the extent practicable.

In summary, we have conducted a case study applying quantitative, comparative risk analysis to assess population health benefits and risks of various remediation scenarios for the Hudson River PCBs Superfund Site. This analysis included a range of human health impacts not typically considered within U.S. EPA's risk-assessment and decision-making frameworks but are of a magnitude that warrants consideration. Similar comparative risk analyses should be conducted on other large sites to evaluate the usefulness of our approach. Overall, our work suggests that use of comparative risk analysis in population-based assessments of environmental remediation alternatives has the potential to better inform cost-effective decision-making that maximizes benefits while minimizing risks to human health.

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